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Modern Concepts of Cardiovascular Disease

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THE CLINICAL IMPORTANCE OF THE VARIOUS FACTORS DETERMINING CORONARY BLOOD FLOW*

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The heart, unlike other muscles in the body, has no long periods of rest. It is continually beating, and to continue to do so, it must completely restore the chemical sources of energy which it loses in systole during the succeeding diastole. It is imperative, therefore, that an adequate and flexible blood supply be provided for this incessantly active organ. This is the function of the coronary circulation, interference with which may prove rapidly fatal.

The coronary circulatory system has a number of peculiarities which distinguish it from other circulatory systems in the body. It is the first branching which comes off the line; it feeds the pump itself, and it is enclosed in part by the constantly active muscle walls of the heart.

The coronary system can be divided into the following divisions:

A. *The coronary arteries, the irrigating tubes.* These are further divided into *superficial* and *intramural* branches because there are differences in the blood flow in these subdivisions.

B. *The capillaries.* These serve here as elsewhere for the interchange with the tissue cells except for the small interchange which occurs via the endothelial lining of the cavities of the heart with its many sinusoidal impouchings.

C. *The drainage system.* This consists of the *intramural* and *superficial veins*, the *coronary sinus* and the *Thebesian channels.* While these drainage channels under normal circumstances act only to drain away the blood, they can act as irrigating channels to the capillaries under certain circumstances when the pressure gradients are reversed. Reversal of flow in the drainage system is encountered in closure of the coronary arteries. The practical utility of this reversal of flow is somewhat limited, however, since experience has shown that the majority of exits in this drainage system (90% or more) are in the right side of the heart. Little can be expected from these channels in making up a deficit of blood in the capillaries of the wall of the left ventricle. It is no doubt this peculiar circumstance that makes infarction of the auricles and of the right ventricle so rare and infarction of the left ventricle under like circumstances so common.

The coronary arteries are not perfect end arteries as was considered to be the case at one time. With advance in age, intercommunications constantly increase and serve as collaterals in case of need. Under similar circumstances extracardiac anastomotic communications also can develop. The current belief that the partition of blood flow in the coronary system is fixed within narrow limits both as regards the irrigating and drainage tubes is not substantiated by experience. Actual observation has shown that the partition of blood flow through the different coronary irrigating channels is variable and so is the partition of blood from the various feeding arteries through the various channels for drainage.

There are a number of ways of studying the dynamics of the coronary circulation in animals, although in man we must judge from clinical and electrocardiographic disturbances. In animals the fluctuation in pressure in the various vessels can be recorded and measured. The pressure gradients can be established, but it is hazardous to judge the nature of the blood flow from these alone as other circumstances play an important role. Flow measurements should be made directly in terms of *volume flow.* Linear velocity measurements are not sufficient since the caliber of the coronary vessels is variable and volume flow is the product of linear velocity and cross section area. Such changes in caliber occur not only over long periods of time but also during the cardiac cycle. Furthermore, a distinction must be made between flow in a given vessel and total coronary flow. For example, too much of our information concerning the action of various drugs, hormones and other physiological stimuli on the total coronary flow has depended upon the measurements of the outflow from the cannulated coronary sinus. Recent experience has shown that changes in coronary sinus flow may be the result of redistribution of blood between the coronary sinus and the Thebesian channel drainage and not at all a measure of the total flow in the coronary capillaries. The only certain way of determining the flow in the coronary capillaries we have found is to measure the total inflow into the coronary arteries.

*No attempt need here be made to present the nature of the variation of flow during the cardiac cycle in the various parts of the coronary system because while interesting in itself, it does not greatly advance our knowledge of the adaptibility of the circulation of coronary blood from the practical standpoint.

When one evaluates the work on coronary flow, with these principles in mind, certain fundamental facts stand out. The few seeming paradoxes and sharp differences in observations can be reconciled for the most part by applying the principles outlined above.

In brief, the coronary blood flow is determined by the balance of action of a number of factors:

1. The driving force existing at the mouths of the coronary arteries and set by the pressure in the aorta is one of the most important. This is to be considered not as the mean pressure but as the cyclic fluctuations in pressure which can be measured accurately in man. While the pressure existing in the aorta in diastole is the more important, the pressure during systole cannot be ignored.

2. The resistance to flow of blood is another important factor. This is determined in part by changes in the caliber of the various subdivisions of the coronary vessels and in part by the pressure existing at the exits of the drainage channels, namely the pressures in the right auricle and ventricle. The caliber of the coronary vessels is mainly determined by the changes in shape and stress in the walls of the heart which are the extravascular supports of these vessels. To a much smaller extent, the caliber is determined by the internal pressure in these vessels transmitted from the aorta or backward from the right auricle and ventricle. Active changes in the muscular coats of the vessels play a minor role in determining the coronary vessel caliber. There has been no clear evidence that coronary spasm does or can occur. Too little attention has been paid to the important part played by the extravascular support. Recent experience has shown that this is probably at least as important as the pressure in the aorta in determining coronary flow. The coronary blood vessels unlike vessels in noncontractile organs go through rapid passive changes in internal tension, in caliber and in shape because of the action of the heart chambers. The result of this action of the heart walls is twofold. On the one hand it tends to act as a pump propelling blood onward, but in addition it tends to compress the vessels and decrease their caliber. The balance of the two actions varies in the different chambers of the heart and in different parts of the wall of the same chamber: it alters greatly under different dynamic circumstances. Too often the action of various procedures has been ascribed to active changes in the wall of the coronary vessels, when in reality it was due entirely to passive changes following alteration of this extravascular supporting mechanism.

We have recently studied this particular problem and found that the net result of increasing the power of the heart when the aortic pressure is kept constant is to decrease coronary capillary flow. Changes in heart rate at constant heart power under the same conditions had little effect on the coronary capillary flow. An investigation was also made of the active changes in the coronary vessels after eliminating this extravascular action. The caliber of the coronary vessels we found to undergo but little active change. Nitrites cause a noticeable dilatation, the xanthine derivates but relatively little. Acetylcholine and mecholyl cause dilatation. Adrenaline usually causes dilatation, but a preliminary constriction is not uncommon. Pitressin, digitalis, and apparently histamine, cause constriction. With the exception of pitressin, none of these are powerful agents. They are decidedly less powerful than either anoxemia or carbon dioxide excess which are potent dilators.

As regards the innervation of the coronary vessels, we have found, contrary to the generally accepted view, that the nerve control is not very powerful and that the action of the nerves is no different than in other parts of the body. The parasympathetics, the vagi, exert a small tonic dilator action, while the sympathetic nerves have a small tonic dilator and constrictor action, the latter being the more powerful. There is nothing in our studies to suggest that these nerves are potent elements in the control of the coronary blood flow.

In other words, our studies indicate that active changes in the muscle wall of the coronary arteries are practically of little significance in comparison with the other factors controlling flow except for asphyxia. Since all instances of asphyxia of the heart tend to produce coronary dilatation, this becomes an important compensatory mechanism in times of stress. The important factors involved in determining coronary flow are on the one hand the contracting heart itself and on the other the pressure level in the aorta.

The mere fact that under normal circumstances the heart can go through a tremendous range of effort without deleterious influence shows that in the normal individual the coronary flow is adjusted by these two factors to keep pace with the work that the heart does. Surprise should not be expressed that the coronary flow occasionally fails to do so, but rather that it does not do so more often. In clinical practice we are not so much concerned with the absolute quantity of blood reaching the heart nor the absolute rate of delivery, as with the quantity and rate of the blood flow in the coronary capillaries in relation to the needs of the heart at the moment to restore the energy which the cost of its work demands. When this balance cannot be maintained because of occluding diseases of the coronary arteries, or a lack of maintenance of the aortic pressure, particularly in diastole, or a poor quality of the blood itself, then definite symptoms and signs develop which constitute the various clinical syndromes of coronary insufficiency. In the presence of coronary disease, any abrupt increase in the work of the heart or any abrupt drop in pressure in the aorta may quickly establish a vicious circle which suddenly causes acute coronary insufficiency and gives the spasmodic character to the clinical picture. Every instance of acute coronary insufficiency can be adequately accounted for in this way without recourse to so-called coronary spasm.

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